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The relation of environmental tobacco smoke (ETS) to chronic bronchitis and mortality over two decades☆

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ABSTRACT

Introduction: Our aim was to describe how the prevalence of subjects exposed to environmental tobacco smoke (ETS) has changed from 1992 to 2012 in Finland. We also investigated the association between ETS and chronic bronchitis and cause-specific and all-cause mortality.

Methods: The study population is composed of 38 494 subjects aged 25–74 years who participated in the National FINRISK Study between 1992 and 2012. Each survey included a standardized questionnaire on exposure to ETS, symptoms of chronic bronchitis, smoking habits and other risk factors, and clinical measurements at the study site. Data on mortality was obtained from the National Causes of Death Register.

Results: In 2012, 5% of the participants were exposed to ETS compared to 25% in 1992. The adjusted odds ratio (OR) for ETS exposure in 2012 compared with that in 1992 was 0.27, $p < 0.001$. Exposure to ETS was more common in men than in women and among smokers than in non-smokers. Exposure to ETS was in turn associated with chronic bronchitis, OR 1.63 (95% confidence interval 1.49–1.78), – also separately both at work (OR 1.36) and at home (OR 1.69). Subjects with exposure to ETS had significantly increased all-cause (hazard ratio = HR 1.15, 1.05–1.26) and cardiovascular mortality (HR 1.26, 1.07–1.47). However, when stratified by smoking ETS was associated with all-cause mortality only in smokers (HR 1.31, 1.15–1.48).

Conclusion: The proportion of subjects exposed to ETS decreased substantially during the study. Additionally, ETS exposure was associated with chronic bronchitis throughout the study and increased all-cause and cardiovascular mortality.

1. Introduction

Environmental tobacco smoke (ETS) originates from tobacco combustion [1]. ETS is a major indoor air pollutant when there is smoking inside [1,2]. Furthermore, indoor air pollution matters because people can spend up to 90% of their time indoors [2]. Inhalation of tobacco smoke causes airway inflammation and enlargement of the mucous glands, leading to increased phlegm production [3] and chronic bronchitis [4]. The main risk factor for chronic bronchitis is active smoking [4], but exposure to occupational airborne particles, biomass burning, chest infections in childhood and ETS also play a role in its pathogenesis [5–9].

In the FINRISK Study in Finland, smoking has decreased in men during 1982 and 2007 [5]. In another study in nine European countries (including also Finnish cohorts) [10], smoking has declined between 1985 and 2000 among men from all educational levels and among the tertiary and higher secondary educated women. Smoking rates have also decreased in the US population [11]. Parallel to the decrease in smoking, during recent decades in Finland, the prevalence of chronic bronchitis declined from 19% to 13% and 13% to 11%, respectively, in men and women [5]. In Sweden, particularly symptoms of bronchitis decreased in conjunction with a 30% decrease in the prevalence of smoking [12]. Chronic bronchitis can in turn lead to the development of COPD (chronic obstructive pulmonary disease) [13]. With decreasing smoking, the prevalence of COPD has also decreased or been stable during the same time period [14–16]. The decrease in smoking

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Table 1
Description of the study population by the year of examination.

n	The year of examination				
	1992	1997	2002	2007	2012
Invited	7927	11 500	13 498	12 000	10 000
Examined (men/women)	2849/3202	4253/4193	4482/5098	3740/4253	3041/3383
Non-responders (men/women)	1116/760	1747/1307	2267/1651	2260/1747	1959/1617
Smoking status					
never smokers	2596	3752	4294	3634	3034
ex-smokers	1622	2317	2490	2425	1962
current smokers	1811	2117	2725	1855	1345
excluded[*]	22	260	71	79	83
Chronic bronchitis[†]	855	1085	1175	879	686
Exposure to environmental tobacco smoke (= ETS)[‡] (%)	25.0	12.6	11.6	10.1	5.0
Mean hours/day (SD)[§]in exposed	4.98 (5.32)	4.63 (5.45)	5.13 (5.70)	3.93 (5.13)	7.11 (8.03)
Exposure to ETS at work/at home/other[‡] (%)	–	–	5.4/3.4/4.2	4.0/2.2/5.0	2.3/2.1/1.9

[*]Excluded were subjects without data on smoking or when last smoked and ex-smokers who had quit smoking less than one month ago.
[†]In subjects having data of smoking.
[‡] Self-reported exposure to environmental smoke at each examination in subjects having data of smoking habits.
[§]In subjects exposed to ETS and having data on smoking habits. SD = standard deviation.

prevalence has also led to a reduction in ETS [5,12]. In altogether 26 European centers, the prevalence of individuals exposed to ETS decreased from 38.7% in 1990–1994 to 7.1% in 2008–2011 [17].

ETS exposure has also been associated with increased mortality [18]. In never-smoking women, ETS exposure at home has been associated with increased all-cause mortality (hazard ratio = [HR] 1.15) and cardiovascular mortality (HR 1.37), and ETS exposure at work has been associated with increased cancer mortality (HR 1.19)[18]. The present study describes how the prevalence of subjects exposed to ETS has changed from 1992 to 2012. We also examined the association between ETS and chronic bronchitis and cause-specific and all-cause mortality stratified by smoking.

2. Methods

2.1. Sample

The study population consists of 38 494 subjects aged 25–74 years who participated in five cross-sectional surveys of the National FINRISK Study between 1992 and 2012 carried out by the National Institute for Health and Welfare (Table 1). At each survey [5,19], an independent random population sample was taken from the population register in six geographical areas: the provinces of North Karelia and Kuopio in eastern Finland since 1972, the Turku-Loimaa area in south-western Finland since 1982, the capital area (the cities of Helsinki and Vantaa) since 1992 and the provinces of Oulu (since 1997) and Lapland (since 2002) in northern Finland. The sampling and study methods complied with the protocol of the World Health Organisation MONICA (Multinational MONItoring of trends and determinants in CArdiovascular disease) project [20] and since 2002, the later recommendations of the European Health Risk Monitoring Project (EHRM) [21].

2.2. Questionnaire

At each examination, the participants received a self-administered questionnaire [5]. Exposure to environmental tobacco smoke (ETS) was determined by the question 'How many hours do you daily spend in indoor spaces where you have to inhale other people's smoke?' Since 2002 onwards exposure to ETS separately at work, at home and at other places was enquired. The diagnosis of chronic bronchitis was based on a positive response to the same standard question 'Do you bring up phlegm on most days or nights for at least three months each year' [5,13,22] in the questionnaire. Smoking, education, occupation, marital status, a history of hypertension during the preceding 12 months, a history of diagnosed myocardial infarction and self-reported asthma were asked with standardized questions in the questionnaire. Body mass index (BMI) (kg/m^2) was calculated from height and weight measured at the examination.

2.3. Definitions

For the present study, exposure to ETS was first classified as a binary variable (no/yes). Between 2002 and 2012, exposure to ETS was additionally classified into five categories: no exposure, exposure only at work, only at home, only at other places and multiple exposure (if a subject had exposure to ETS e.g. both at work and at home). Smoking status was classified into the three categories: never-, ex- and current smokers (Table 1). Smokers had smoked regularly at least one year. Ex-smokers had stopped smoking at least one month before the survey. Excluded from analyses (altogether n = 515) were those who had no complete data on smoking and those smokers who had quit smoking less than a month ago (thus could not be classified into ex-smokers because of their short period of abstinence) [5].

The level of education was classified into four categories: elementary school, vocational school, upper secondary school or college and an academic degree. Occupation was graded into seven categories: agriculture and dairy farming; factory, mine and construction work; office work; unemployed; students; housewives and pensioners. Marital status was broken down into four categories: married or cohabitation without marriage, unmarried, divorced and widowed. The area of residence was categorized into four classes: North Karelia and Kuopio, the Turku-Loimaa area, Lapland and Oulu, and the capital area.

2.4. Assessment of mortality

Data on mortality was obtained from the National Causes of Death Register. Between 1992 and 2012 two different revisions of *the International Classification of Diseases* (*ICD*) were used in Finland –the *ICD-9* between 1987 and 1995 and the *ICD-10* since 1996. In the present study, a concordance table was used for bridging the two revisions of ICD [23], and the underlying causes of death were classified into the following four major categories: respiratory causes, cardiovascular diseases, cancer and other causes. The listing of the corresponding *ICD-9* and *ICD-10* codes has been shown earlier [24].

2.5. Statistical methods

Logistic regression analysis was used to study age-specific prevalence odds ratios (ORs) for exposure to ETS (n = 36 594) during 1992 and 2012. Two models were fitted. The first model included the following variables: the survey year (as a continuous variable), smoking status and the age –group (as categorical variables); the second model additionally included the following categorical variables: sex, occupation, education and the area of residence. The results are presented as odds ratios with 95% confidence intervals (95% CI). A significance level of $p < 0.05$ was used.

Multivariable logistic regression analysis was used to examine the association between exposure to ETS and chronic bronchitis (n = 34 101). Those who had no data on the symptoms of chronic bronchitis or on self-reported asthma and subjects who reported having asthma diagnosed by a doctor were excluded. A separate multivariable logistic regression analysis was done from 2002 onwards to study the relationship of ETS exposure at work, at home and at other places to chronic bronchitis (n = 20 932). In smokers, additional adjustment for pack-years was done as a continuous variable (pack-years = years of smoking x the daily number of cigarettes smoked/20).

ANCOVA analysis was used to compare mean hours of ETS exposure by smoking status and at work, home and other places. These analyses were adjusted for sex, the age group, the survey year, smoking status and the area of residence.

Cox proportional hazards regression models were used to analyse the relation between ETS exposure and all-cause and cause-specific mortality. Among subjects with variables needed for multivariate analyses 506 subjects had participated in the FINRISK study twice between 1992 and 2012. Among these subjects, the values of the first survey were used in the mortality analyses. The mortality analyses (n = 33 266) were first adjusted for the age group and smoking status and then additionally adjusted for sex, education, marital status, the area of residence, a history of myocardial infarction and the presence of hypertension as categorical variables, and the survey year and body mass index as continuous variables. In smokers, the mortality analysis was additionally adjusted for pack-years. The results are presented as hazard ratios with 95% confidence intervals. A significance level of $p < 0.05$ was used.

Statistical analyses were performed with SPSS 22 for Windows. All surveys had ethics approval from the ethics committees operating at the time of data collection in 2001 and by the ethics committee at the National Institute for Health and Welfare in Helsinki, and participants have given their informed consent.

3. Results

Between 1992 and 2012 the percentage of subjects who were exposed to ETS decreased from 25% to 5% (Table 1). Between 2002 and 2012 the percentage of subjects who were exposed to ETS at work, at home and at other places also decreased (Table 1). However, in subjects exposed to ETS, the mean hours spent daily in ETS exposure were highest in 2012 (Table 1).

In logistic regression (Table 2), there was a significant ($p < 0.001$) decreasing trend in exposure to ETS between 1992 and 2012 (OR 0.933). After inclusion other covariates into the Model 2, the ORs decreased only slightly. In Model 2, the adjusted OR for exposure to ETS in 2012 compared to that in 1992 was 0.27 ($=0.936^{20}$). There was also an interaction ($p < 0.001$) between the age group and the decreasing yearly trend, so that the decreasing trend was insignificant in the oldest age group (Table 2). Generally, exposure to ETS was significantly more common in men than in women (OR 1.76 (95% CI 1.64–1.89)) and in smokers than in never smokers (OR 2.29 (95% CI 2.12–2.48)) (not shown).

ETS exposure was in turn associated with chronic bronchitis (OR 1.63) (Table 3). Stratified by smoking (Table 3), ETS exposure was associated with chronic bronchitis in smokers (OR 1.75 (95% CI 1.55–1.97)). ETS exposure was related to chronic bronchitis in ex-smokers and never smokers too (OR 1.47 (95% CI 1.20–1.81) and 1.48 (95% CI 1.24–1.76), respectively) although ETS exposure was less pronounced ($p < 0.001$) in ex-smokers and never smokers than in smokers (Table 3). In smokers, exposure to ETS was still significantly associated with chronic bronchitis after additional adjusting for pack-years (OR 1.53 (95% CI 1.35–1.73)) (not shown). Generally, there were no significant interactions between ETS exposure and the survey year, sex, the age group or occupation on the presence of chronic bronchitis.

ETS exposure separately at work, at home and at other places was significantly associated with chronic bronchitis (Table 4), and the mean daily hours spent in ETS exposure were significantly higher at home than at work. Stratified by the smoking group (not shown), ETS exposure at work and at other places was significantly associated with chronic bronchitis in smokers (OR 1.51 (95% CI 1.14–2.02) and 1.58 (95% CI 1.19–2.10), respectively). ETS exposure at home was significantly associated with chronic bronchitis both in smokers and never smokers (OR 1.68 (95% CI 1.26–2.24) and 2.03 (95% CI 1.20–3.42), respectively). In smokers, after additional adjusting for the pack-years ETS exposure at work was still associated with chronic bronchitis with OR 1.39 (95% CI 1.02–1.87).

ETS exposure was associated with increased mortality (Table 5). In Model 1, after adjusting for the age group and smoking status HRs (=hazard ratios) for all-cause and cardiovascular mortality were 1.25 and 1.37, respectively, and the HRs decreased after a multivariable adjustment in Model 2. When the analysis was performed separately in each smoking group (Table 5) ETS exposure was associated with increased mortality only among smokers. In smokers, after additional adjusting for pack-years HRs for all-cause mortality were 1.18 (1.04–1.35, p = 0.011) and 1.14 (1.00–1.30, p = 0.058) in Model 1 and 2, respectively. After including chronic bronchitis into the Model 2, there was an interaction between ETS and chronic bronchitis on mortality in men (p = 0.035) so that HRs for all-cause mortality in men with chronic bronchitis without and with ETS exposure were 1.25 (1.11–1.41) and 1.55 (1.29–1.85), respectively, compared with men without chronic bronchitis or ETS exposure.

Fig. 1 illustrates the effect of ETS exposure on survival during the study. In Fig. 1 (adjusted for the age group and smoking status, Model 1), the HRs for all-cause mortality were 1.13 (0.99–1.29, p = 0.063) and 1.36 (1.21–1.52, $p < 0.001$), respectively, in subjects with exposure to ETS < 2 h/day and ≥ 2 h/day. In Model 2 (not shown in Fig. 1), the corresponding HRs were 1.00 (0.88–1.14, p = 0.980) and 1.29 (1.15–1.46, $p < 0.001$), respectively.

4. Discussion

In the present study, the proportion of subjects exposed to ETS decreased clearly during the last decades. ETS exposure was significantly associated with chronic bronchitis in all smoking groups. ETS exposure was also associated with increased all-cause and cardiovascular mortality. But when mortality analysis was stratified by smoking status there was an increased risk for all-cause mortality only in smokers.

In the present study, the prevalence of subjects exposed to ETS in 2012 was 5%, which is consistent with another European study [17]. With the reduction in smoking prevalence and policies to restrict smoking in public places ETS exposure has been decreasing both in Europe and in the United States [17,25]. In Finland, the first law to restrict smoking was put into operation in 1977 [26], and since the 1980s' ETS exposure has decreased significantly [5]. The influence of enforcements of the tobacco laws restricting smoking at worksites in 1995 and banning of smoking in bars and restaurants in 2007 [27] can be seen also in this study as clear decreases in exposure to ETS between 1992 and 1995 and between 2007 and 2012. Between 1985 and 2000 in Finland, ETS exposure at work has been three times more common in smokers than in non-smokers, but ETS exposure has decreased both

Table 2
Adjusted odds ratios (OR) for the trend in exposure to environmental tobacco smoke (ETS) during 1992–2012 by age group.

	n	Model 1*		Model 2†	*p-value*
		OR‡ (95% CI) for 1992–2012	*p-value*	OR‡ (95% CI) for 1992–2012	
Age group					
25–34	7043	0.931 (0.921–0.942)	*< 0.001*	0.938 (0.927–0.949)	*< 0.001*
35–44	7760	0.918 (0.908–0.928)	*< 0.001*	0.921 (0.911–0.932)	*< 0.001*
45–54	8190	0.924 (0.915–0.934)	*< 0.001*	0.925 (0.915–0.936)	*< 0.001*
55–65	8581	0.951 (0.941–0.962)	*< 0.001*	0.950 (0.939–0.962)	*< 0.001*
65–74	5020	0.986 (0.964–1.009)	*0.230*	0.998 (0.974–1.023)	*0.871*
All	36 594	0.933 (0.928–0.938)	*< 0.001*	0.936 (0.931–0.942)	*< 0.001*

*From a logistic regression model adjusted for smoking status and age-group. 95% CI = 95% confidence interval.
†From a logistic regression model adjusted for smoking status, age-group, gender, occupation, education and area of residence.
‡OR is presenting the yearly change in exposure to environmental tobacco smoke.

Table 3
Multivariable adjusted odds ratios (OR) for chronic bronchitis during 1992–2012 by exposure to environmental tobacco smoke (ETS).

Exposure to ETS	n	mean hours of ETS/day (SD*)	OR (95% CI†) for chronic bronchitis	
All‡ (n = 34 101)				*p-value*
Exposed to ETS	4432	4.84 (5.58)	1.63 (1.49–1.78)	*< 0.001*
Exposure to ETS, mean hours/day				
0 < & < 2	2368	1.25 (0.45)	1.52 (1.36–1.71)	*< 0.001*
≥2	2064	8.96 (5.90)	1.74 (1.55–1.95)	*< 0.001*
Smoking group§				
Never-smokers (n = 15 454)				
Exposed to ETS	1329	4.12 (5.05)	1.48 (1.24–1.76)	*< 0.001*
Ex-smokers (n = 9713)				
Exposed to ETS	1003	3.84 (4.56)	1.47 (1.20–1.81)	*< 0.001*
Smokers (n = 8934)				
Exposed to ETS	2100	5.78 (6.16)	1.75 (1.55–1.97)	*< 0.001*

*Standard deviation.
†95% CI = 95% confidence interval.
‡From a logistic regression model adjusted for gender, survey year, age group, smoking status, occupation, education and area of residence. Subjects without ETS exposure as a reference group.
§From a logistic regression model adjusted for gender, survey year, age group, occupation, education and area of residence. For each smoking category separately. Subjects without ETS exposure as a reference group.

among never smokers and smokers [28]. In the present study, ETS exposure was still more frequent in smokers than in never smokers and in men than in women.

In the present study, exposure to ETS was associated with chronic bronchitis, also separately both at work and at home. Among non-smokers, chronic bronchitis has been associated with the number of smokers at work [29]. Living with smokers before the age of 18 has associated with chronic phlegm production in adulthood [8]. In female never smokers, ETS exposure at work has a stronger association with COPD than ETS exposure at home [7]. In another study, the risk of developing COPD has been 1.60 and 1.68 times higher in the highest quartile of ETS exposure in the workplace and at home, respectively, after controlling for smoking history and other factors [30].

It has been proposed, that exposure to the particle phase rather than the vapour phase of exhaled cigarette smoke is associated with chronic bronchitis in passive smoking [9]. It is not known how low levels of ETS adversely effects the airways. Additionally, ETS can be also derived from e-cigarettes, though at lower levels than from conventional cigarettes [31]. In the present study, according to the odds ratios ETS exposure was more strongly associated with chronic bronchitis in smokers than in never and ex-smokers, but smokers had the highest mean daily ETS exposure. In Switzerland, an increasing risk for chronic bronchitis with increasing daily hours of ETS exposure has also been found [9].

The risk of chronic bronchitis has doubled if there has also been occupational exposure to biological dusts [6]. However, in one study, there was no evidence that occupational exposure to gas and dusts modified the association between ETS and COPD [30]. In the present study, there was no significant interaction between ETS and occupation on chronic bronchitis. Our classification of occupations into the seven categories gave only a rough estimate of occupational exposures, however, and the classification was done only once at each survey. According to our earlier results, male smoking and the prevalence of chronic bronchitis in Finland has been decreasing [5], which is in line with the present results of decreasing ETS exposure. In the present

Table 4
Multivariable adjusted odds ratios (OR) for chronic bronchitis during 2002–2012 by environmental tobacco smoke (ETS) at work, at home and other places.

Exposure to ETS	n	mean hours of ETS/day (SD*)	*p-value*†	OR (95% CI*) for chronic bronchitis (exam 2002/2007/2012)	*p-value*‡
No exposure to ETS	18 926	–		1	*ref*
At work (only)	726	3.87 (3.58)	*ref*	1.36 (1.10–1.69)	*0.005*
At home (only)	405	8.66 (7.51)	*< 0.001*	1.69 (1.34–2.14)	*< 0.001*
At other places (only)	642	1.75 (1.82)	*< 0.001*	1.56 (1.27–1.92)	*< 0.001*
Combination (e.g. at work + at home)	233	10.24 (8.27)	*< 0.001*	2.60 (1.94–3.50)	*< 0.001*

*SD = standard deviation. 95% CI = 95% confidence interval.
†From ANCOVA analysis adjusted for gender, survey year, age group, smoking status and area of residence.
‡From a logistic regression model adjusted for gender, survey year, age group, smoking status, occupation, education and area of residence.

Table 5
Adjusted hazards ratios* (HR) for all-cause and cause-specific mortality during 1992–2015 by exposure to environmental tobacco smoke (ETS).

Cause of death	No exposure to ETS	Exposure to ETS	Model 1*		Model 2†	
	n of deaths/n in analysis					
All-causes						
All	2907/28 954	594/4312	1.25 (1.14–1.37)	*< 0.001*	1.15 (1.05–1.26)	*0.004*
Never-smokers	1113/13 859	109/1293	1.04 (0.85–1.27)	*0.711*	0.94 (0.77–1.15)	*0.523*
Ex-smokers	964/8561	106/975	1.12 (0.91–1.37)	*0.298*	1.03 (0.83–1.26)	*0.806*
Smokers	830/6534	379/2044	1.42 (1.25–1.60)	*< 0.001*	1.31 (1.15–1.48)	*< 0.001*
Cause-specific causes	n in analysis = 28 954	n in analysis = 4312				
Respiratory diseases	125	31	1.37 (0.91–2.07)	*0.131*	1.29 (0.85–1.95)	*0.235*
Cardiovascular disease	1007	206	1.37 (1.17–1.60)	*< 0.001*	1.26 (1.07–1.47)	*0.005*
Cancer	862	151	1.04 (0.87–1.24)	*0.675*	0.99 (0.82–1.18)	*0.893*
Other	913	206	1.31 (1.12–1.53)	*0.001*	1.19 (1.01–1.40)	*0.034*

*from Cox proportional hazards regression model. Adjusted for age group and smoking status. Subjects without exposure to ETS as a reference group. In cause-specific mortality for each cause separately. 95% CI = 95% confidence interval. †from Cox proportional hazards regression model. Adjusted for age group, smoking status, gender, survey year, education, marital status, body mass index, history of myocardial infarction, presence of hypertension and the area of residence. Subjects without exposure to ETS as a reference group. In cause-specific mortality for each cause separately.

study, there was no significant interaction between ETS and the survey year on chronic bronchitis. Thus, ETS exposure was similarly associated with chronic bronchitis throughout the study.

In the present study, the clear association between ETS exposure and mortality among smokers may indicate that perhaps simultaneous active and passive smoking can have detrimental additive effects on health. In the present study, chronic bronchitis strengthened the effect of ETS on mortality in men. Additionally, in subjects with ETS exposure ≥2 h/day the association between ETS and mortality was stronger. Worldwide approximately 1.0% of all deaths among non-smokers have been estimated to be caused by environmental tobacco smoke [32]. ETS exposure in non-smokers has also increased mortality from lung cancer and COPD [33,34]. Additionally, ETS has been associated with a greater burden of cardiovascular risk factors [35], such as higher BMI and fasting glucose [36], and with endothelial dysfunction and increased plasma fibrinogen levels [37].

The strength of our study was a large study population and a long follow-up. The participation rates decreased during the follow-up, probably leaving out more of those with higher morbidity than healthier persons [5]. According to an earlier questionnaire study, however, non-responders evaluated by a telephone interview have not reported more respiratory symptoms than responders [38]. In addition, probably the response rates did not differ by ETS exposure. Earlier, a high validity of self-reported exposure to ETS [39] has been found, though a change in the exposure pattern may have happened during the course of the present study.

In mortality analyses, there was only the baseline measurement of smoking and the other variables. We know that a large proportion of smokers have stopped smoking during the follow up [19]. However, misclassification of smokers and ex-smokers would only weaken the observed association between ETS and mortality. Unfortunately, pulmonary functions were not measured, and thus we could not study the association between ETS and COPD. There was also no biological marker for exposure to smoke like measurements of cotinine in the blood [40].

In conclusion, exposure to ETS has decreased during the study period. The decrease in ETS may be a result of decreasing smoking prevalence and restrictions of smoking in public places. In the present study, exposure to ETS was associated with an increased risk for chronic bronchitis and elevated cardiovascular and all-cause mortality.

Conflicts of interest

The material has not been published and is not under consideration for publication elsewhere. No author has any conflict of interest. We all have read the paper and approved the submission as well as approved

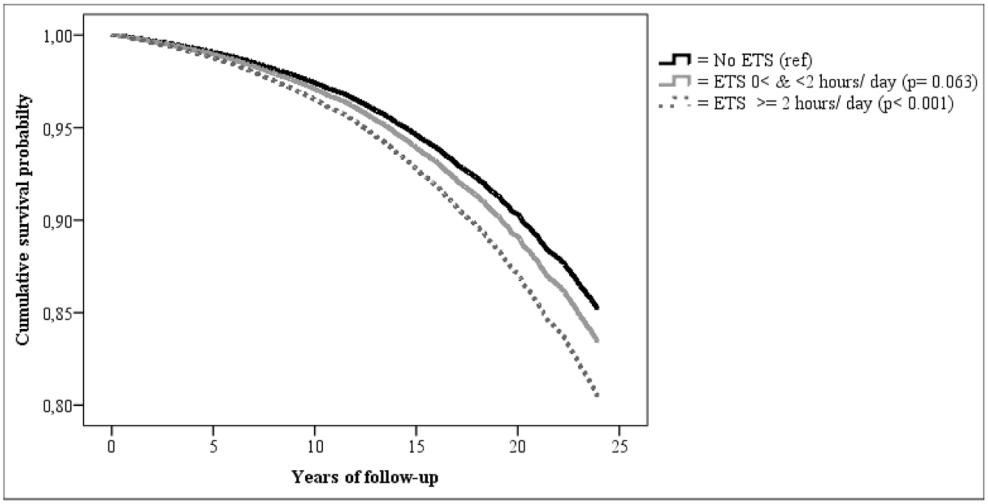

Fig. 1. Cumulative survival probability curves by exposure to environmental tobacco smoke (ETS) based on Cox's proportional hazards regression model, adjusted for age group and smoking status. No ETS as a reference group. N of deaths (n in analysis) 2907 (28 954), 254 (2289) and 340 (2023), respectively, among subjects with no ETS, with ETS 0 < & < 2 h/day and with ETS ≥ 2 h/day. The HRs (=hazard ratios) for all-cause mortality were 1.13 (0.99–1.29) and 1.36 (1.21–1.52), respectively, among subjects with ETS 0 < & < 2 h/day and with ETS ≥ 2 h/day compared with subjects with no ETS.

our names in this paper and thus sign consent to publication.

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